Evaluation of pacing for heart block in myocardial infarction

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A series of 58 patients treated by endocardial pacing for second and third degree heart block complicating acute myocardial infarction is analysed. Forty-five of the patients had complete heart block and 13 had second degree block. The overall mortality was 27.6 per cent.

The very poor prognosis in patients with anterior infarction is confirmed. There were 13 critically ill patients who derived benefit from pacing and survived. Of this group, 11 had had inferior infarctions and would probably have died without pacing. Their survival is reflected in the relatively low mortality of 20 per cent in the whole inferior infarction group. We consider therefore that inferior infarction with heart block, without treatment by pacing, may carry a serious prognosis and that a less active policy of management is not justified.

Severe haemodynamic deterioration occurred in all 8 patients over 70 years, but cardiogenic shock was reversed by pacing in 5 survivors, indicating that an aggressive approach to treatment of heart block in the elderly is warranted.

Analysis of the deaths does not suggest that routine lignocaine infusions would have reduced the mortality.

Complete heart block occurs as a complication of myocardial infarction in 2.5 to 8 per cent of cases, and carries a mortality of 50-60 per cent. Second degree heart block is reported to occur in 4.6 per cent of cases with a mortality of 48.3 per cent (Cross, 1968). There is still considerable controversy over the value of pacing, and while most coronary care units probably pace patients developing second and third degree heart block, the mortality rates in several reported series remain high, i.e. Lassers and Julian (1968) 48 per cent; Sutton, Chatterjee, and Leatham (1968) 45.5 per cent; and Cross (1968) 51 per cent. However, Bouvrain (1968) reports a drop in mortality from 60 to 32 per cent. Doubts have been expressed as to whether all patients with second and third degree heart block should be paced (Norris, 1969; Friedberg, Cohen, and Donoso, 1968). Norris suggests that patients with heart block complicating inferior infarction do not require routine pacing because of their good prognosis. In an attempt to evaluate cardiac pacing and determine the prognostic points and indications for pacing in acute heart block complicating myocardial infarction, we have analysed a series of 58 cases treated at the Groby Road Hospital, Leicester, by endocardial pacing.

Subjects and methods

The series consists of 58 consecutive cases of second and third degree heart block complicating myocardial infarction admitted to the Cardiac Unit of the Leicester Regional Cardiothoracic Unit at Groby Road Hospital, Leicester, over a 4-year period up to June 1969. Sixteen of these patients had been admitted directly to the unit as cases of myocardial infarction, and were noted to be in or subsequently to develop heart block. Forty-two of the patients were transferred from the Leicester Royal Infirmary and the Leicester General Hospital for pacing after the development of heart block. The patients comprised 13 cases of second degree heart block, and 45 cases of third degree heart block. The diagnosis of myocardial infarction was confirmed by the presence of abnormal Q waves accompanied by ST segment changes, and T wave inversion. In those cases of anterior infarction with left bundle-branch block (LBBB) the diagnosis was confirmed by an increase in serum enzymes or by the appearance of typical Q waves following the resolution of the bundle-branch block pattern. Of the 16 patients admitted directly to the unit, heart block was present on arrival in 6. Thus, little information has been obtained regarding preceding conduction disturbances. A separate coronary care unit did

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not exist during this period, the patients being looked after in a general cardiological ward. They were monitored continuously on a bedside electrocardiograph cathode-ray oscilloscope until they had been in stable sinus rhythm for several days or no other indication for monitoring existed. Heart failure was treated with digoxin and diuretics, and oxygen administered when shock, cardiac failure, or disorientation were present. Severely ill patients were frequently treated on oral anticoagulants despite the theoretical risk of perforation of the ventricular wall by the catheter electrode producing tamponade.

All patients were paced endocardially by the transvenous route, a 5F bipolar electrode catheter (USCI C51) or a 5F unipolar catheter (U.S.C.I. C50) being used. It is our opinion that 6F electrode catheters should not be used as the stiffer catheter probably increases the risk of perforation.

The electrode catheter was introduced via the right external jugular vein in the first 17 cases, but subsequently the right median cubital vein or the right subclavian vein (entered by percutaneous puncture via the supra- or infraclavicular routes) has been used. The subclavian route undoubtedly gives a maximum degree of stability, and is of greater convenience to the nursing staff, is much more comfortable for the patient, and is now used preferentially. The catheter electrodes were inserted in all cases under fluoroscopic control in the adjacent cardiac catheterization laboratory. Generally a stimulating threshold below I volt was obtained and pacing started at 70 a minute with a pulse width of 2 m./sec., and a voltage approximately twice that of the stimulating threshold. An attempt was always made, if possible, to pass the catheter to the pulmonary artery in order to avoid the discontinuance of pacing due to inadvertent insertion of the electrode into the coronary sinus. Except for the first six cases, all patients were paced on demand with a Devices Pace Maker (P. J. Reynolds Ltd.). The catheters were normally left in situ for 21 days after the return to sinus rhythm, though the demand pacemaker was left attached to the electrode for 48 hours after the return to sinus rhythm.

Results

The group comprises 48 male and 10 female patients with an age range between 42 to 78 years, and a mean age of 60.3 years. Sixteen patients (mean age 63.3 years) died, and 42 survived, giving a mortality of 27.6 per cent. The age distributions in the whole group and the fatality group are depicted in Table 1.

It was possible to ascertain the approximate period between the onset of chest pain and heart block in 52 patients, the mean being 1.8 days, with a range of o to 7 days. Of the survivors, the duration of heart block was accurately documented in 40 patients, and the mean duration of pacing before the return of sinus rhythm was 5.9 days, with a range of a few hours to 28 days. In 28 of these patients

TABLE I Age distributions in whole group and fatality group

	Age dis	tribution o	of series		Age distribution of deaths					
Age	40-50	51-60	61-70	71-80	40-50	51-60	61-70	71-80		
No.	9	17	24	8	I	5	7	3		

the heart block occurred within 48 hours of the infarction. In those cases in which heart block occurred at a later date, it was frequently preceded by further severe chest pain, suggesting an extension of the infarction. With the exception of 2 patients in whom atrial fibrillation occurred and persisted following the recovery from heart block, all the survivors returned to sinus rhythm, and in only 2 patients did the PR interval exceed 0.2 sec. two months after discharge from hospital.

The electrocardiographic findings and associated mortality are shown in Table 2.

TABLE 2 Electrocardiographic findings and associated mortality

Electrocardiogram		No. of deaths
Anterior with left bundle-branch block		2
Anterior (previous inferior) with left bundle-branch block		0
Anterior with right bundle-branch block		4
Anterior	2	ò
Inferior with left bundle-branch block		0
Inferior with right bundle-branch block		0
Inferior with right bundle-branch block (previous anterior)		I
Inferior (previous anterior)	7	2
Inferior	39	7

It should be noted that of the 9 anterior infarctions 6 died, giving a mortality of 66.6 per cent. Forty-nine patients had inferior infarctions, reflecting the higher incidence of heart block with lesions at this site, and of this group 10 patients died giving a mortality of 20.4 per cent.

The complications from pacing are listed in Table 3.

TABLE 3 Complications of pacing

Local and systemic infection	
Serious arrhythmias during electrode insertion	2
Serious arrhythmias during pacing	I
Repositioning for displacement (7 patients)	10
Phlebitis of arm vein	3
Perforation	3
The state of the s	

No fatalities resulted from the complications. In two patients ventricular fibrillation occurred during impaction of the electrode in the right ventricle. Heart block returned spontaneously after withdrawal of the catheter from the right ventricle in one case, and the other patient required external DC shock to terminate the ventricular fibrillation.

In one patient still requiring a demand pacemaker, ventricular fibrillation occurred unexpectedly after six days. His clinical condition had been satisfactory until then. Before the onset of ventricular fibrillation his own complexes had been inhibiting the pacemaker, and after successful resuscitation he continued to pace satisfactorily, without any evidence of failure of the pacemaker to be inhibited by low intracavity potentials (Chatterjee, Sutton, and Davies, 1968). Right ventricular perforation by the catheter electrode probably occurred in 3 patients. This was manifested by the pacing threshold rising above 5 volts and the occurrence of praecordial chest pain. Pericardial friction rubs were not heard.

Complications of air embolism or pneumothorax have not been encountered with the use of the subclavian route.

Only 6 cases were paced with fixed-rate pacemakers before the introduction of the demand units in 1967. Competition following the return to sinus rhythm was noted in 3 of these patients, without any serious arrhythmia occurring.

Deaths

Sixteen deaths occurred, complete heart block being present in 13 of these patients (Table 4).

TABLE 4 Mortality in paced heart block

	Complete heart block	Second degree heart block	
Total	45	13	
Deaths	13	3	
Mortality (%)	28.8	23.0	

Prognostic findings

From Table 2 it can be seen that complete bundle-branch block was associated with a high mortality, and commonly complicated anterior infarction, with extensive ventricular septal involvement. Failure of pacing to revert cardiogenic shock fairly rapidly indicated a poor prognosis, and was an indication of extensive myocardial damage.

Adams-Stokes episodes occurred in 10 patients of whom 6 died. Of these 10 patients, 6 had anterior infarcts, and 4 of them, all with a complete bundle-branch block pattern, died. In these patients, the Adams-Stokes episodes were the first manifestations of heart block, first or second degree block not having been noted to occur first. The only warning of the impending heart block was widening of the QRS complexes in some cases.

Patients significantly benefited

We have analysed the critically ill patients whose condition appears to have been significantly benefited by pacing, and who survived. Severe haemodynamic deterioration was always associated in this group with the occurrence of complete heart block. Thirteen patients, i.e. 22.4 per cent of the total numbers, or 31 per cent of the survivors form this group. Complete heart block was present on admission to hospital in 9 of these patients. All were critically ill with slow ventricular rates (range 20 to 54 a minute), and 12 were in a state of severe cardiogenic shock with hypotension, peripheral cyanosis, and mental confusion or restlessness, with signs of right or left ventricular failure. The remaining patient in whom the blood pressure was maintained had severe left ventricular failure, and recurrent Adams-Stokes attacks associated with a heart rate of 20 a minute. Three of the patients were having Adams-Stokes attacks.

Cardiogenic shock was rapidly reversed by pacing, but heart failure improved slowly over several days, requiring oxygen, digitalis, and diuretic therapy.

Three of these patients have subsequently died at home between 2 and 6 months after discharge from hospital. Two had sustained previous infarctions, while the third remained in persistent heart failure after extensive infarction. The other 10 patients have been followed up for at least 6 months and remain fully ambulant, leading fairly active and unrestricted lives. Only one patient, aged 77 years, has had a recurrence of heart failure requiring a full antifailure regimen.

Five of these critically ill patients were over 70 years, and it is to be noted therefore that all the cases of heart block in the group 70-78 (see Table 1) were associated with severe haemodynamic deterioration, the other a having died. Pacing will thus frequently be of crucial value in the elderly.

In the critically ill group that survived, 11 had inferior infarctions (I having had an anterior infarction previously), and 2 had anterior infarctions without bundle-branch block. However, all 13 had complete heart block.

Analysis of deaths

We have analysed the deaths to determine whether any were of arrhythmic origin and if any of them could have been prevented by the use of antiarrhythmic drugs.

Seven patients with complete heart block died of cardiogenic shock within 48 hours of admission. Shock was not reversed by pacing in any of these patients, and persisted despite return to sinus rhythm in one patient. Necropsy in 6 of them confirmed extensive recent infarction. The seventh patient had sustained a previous fully documented infarction two years before the fatal episode of infarction.

Two patients with complete heart block and cardiogenic shock were much improved by pacing and returned to sinus rhythm, but death occurred suddenly at 2 and 5 days respectively from myocardial rupture. One patient died as the result of a cerebral embolus after the return of sinus rhythm. Another patient who was being paced for complete heart block with recurrent ventricular standstill died in mechanical asystole with monitoring showing electrical capture. Two patients died while being paced for continuing heart block for 13 and 8 days, respectively. The first had been in continuous cardiac failure and had a left bundle-branch block pattern and died after a cardiac arrest. The second patient had been in continuous severe cardiac failure and died after a severe episode of chest pain suggesting an extension of the infarction.

Three patients died 10, 16, and 17 days, respectively, after the return of sinus rhythm and discontinuance of pacing. In none of them was there any evidence of a return of heart block, one patient dying from intractable cardiac failure, another after 3 cardiac arrests, and the third after a further episode of infarction.

It thus appears that none of the deaths was due to primary arrhythmias and none could have been prevented by the routine use of antiarrhythmic drugs.

Discussion

The prognostic features are essentially those described in previous papers (Lassers and Julian, 1968; Sutton et al., 1968). Acute heart block complicating anterior infarction carries a high mortality, and as the majority of the anterior infarctions were also associated with a bundle-branch block pattern, it is difficult to say whether bundle-branch block in itself in acute heart block with anterior infarction is of serious prognostic significance. Of 7 patients with anterior infarction with bundlebranch block, 6 died, but the 2 patients with anterior infarction without bundle-branch block survived. However, this series is too small to say whether the absence of bundlebranch block in anterior infarction carries a better prognosis. There were only 3 patients with bundle-branch block associated with inferior infarctions, but only I of this group died. However, complete bundle-branch block indicates a more peripheral involvement of the conduction system, and is usually associated with extensive septal infarction. It is possibly significant that no patient in the critically ill group that survived had bundlebranch block.

Norris (1969) noted that all his patients with heart block and anterior infarction had Adams-Stokes attacks, and our experience is similar, 6 of 10 patients with anterior infarcts having such attacks, in comparison with 4 of 49 with inferior infarcts.

Doubts have been raised as to the value of routine pacing of patients with acute heart block complicating myocardial infarction. Friedberg et al. (1968) suggested that the case for routine pacing has not been proven. Different series are difficult to compare because the severity of the cases is probably not comparable. However, Cross (1968), reporting on an incidence of 4.6 per cent of second degree heart block, and 8.2 per cent of third degree heart block in a series of 930 patients, found the mortality rate to be 48.3 per cent in the second degree block group which was unpaced, and 51.3 per cent in the third degree group. Our series contains 13 cases of second degree heart block with a mortality of 23 per cent, so it is suggested that pacing was of value in this group. However, the sum of our 16 deaths and the 13 patients who benefited significantly, and who would very possibly have died without pacing, makes up 50 per cent of the series. This figure approaches the 50 to 60 per cent rate in most estimates of mortality in unpaced cases. It could thus be considered suggestive that the group without severe haemodynamic deterioration might have survived without pacing.

Norris (1969) and Brown, Hunt, and Sloman (1969) suggest that, as anterior and inferior infarctions carry such a different prognosis, a varying approach to treatment be adopted according to the site of the infarct. However, 10 of the 16 deaths, and 11 of the 13 critically ill group who definitely benefited by pacing had inferior infarctions. These figures indicate that despite the lower mortality in the inferior infarction group, such cases will frequently be at considerable risk. There were also 6 patients with inferior infarctions who had started to show clinical deterioration before pacing which immediately improved them, but they have not been included under the group of patients who

definitely benefited, as they were not considered critically ill. Had they not been paced further, deterioration might very possibly have continued.

Chatterjee, Harris, and Leatham (1969) have recently recommended that all paced patients be given lignocaine infusions in order to diminish competition from ectopics. Our policy has been to use lignocaine only when ventricular ectopics indicate it, and the analysis of the deaths does not suggest that any of the patients would have been saved had they been treated routinely with lignocaine infusions.

The results in the group aged over 70 years indicate that the elderly patient may be significantly benefited. Many coronary care units limit their patients to under 70 years of age, and these patients might miss the opportunity of being helped.

Sutton et al. (1968) and Brown et al. (1969) found that patients developing heart block as a complication of a second infarction had a high mortality of 82 per cent and 100 per cent in their respective series. Only 3 of our 9 patients with previous infarctions died, and the mortality in this group is not significantly higher than the overall mortality.

Most workers in coronary care appear to agree that pacing is superior to drug therapy in the treatment of heart block complicating myocardial infarction (Symposium on Acute Myocardial Infarction: 1968). The main source of controversy appears to be which patients should be paced. While shock, Adams-Stokes episodes, and heart failure are absolute indications for pacing, it has been suggested that in the absence of haemodynamic disturbances pacing is unnecessary, an increase in heart rate being achieved at the expense of increased myocardial work and oxygen consumption (Lassers and Julian, 1968). We have not noted any return of or increase in chest pain or other evidence of deterioration as the result of pacing. In addition a slow heart rate is an unstable rhythm, predisposing to ectopic activity. We consider it safest to insert electrode catheters in all

patients with second and third degree heart block, and attach them to a demand pacemaker. Approximately 50 per cent of patients with second degree heart block go into complete heart block (Lown, 1968), and as deterioration may occur suddenly after its occurrence, we consider it preferable to pace all such cases.

References

- Bouvrain, Y. (1968). Discussion. In Acute Myocardial Infarction: Proceedings of a Symposium sponsored by the University of Edinburgh, Sept. 1967, p. 149. Ed. by D. G. Julian and M. F. Oliver. Livingstone, Edinburgh.
- Brown, R. W., Hunt, D., and Sloman, J. G. (1969). The natural history of atrioventricular conduction defects in acute myocardial infarction. American Heart Journal, 78, 460.
- Chatterjee, K., Harris, A., and Leatham, A. (1969). The risk of pacing after infarction, and current recommendations. Lancet, 2, 1061.
- , Sutton, R., and Davies, J. G. (1968). Low intracardiac potentials in myocardial infarction as a cause of failure of inhibition of demand pacemaker. Lancet, 1, 511.
- Cross, E. B. (1968). Coronary Heart Diseases Section, U.S. Public Health Services, Washington. A Cooperative Study, pp. 41-42. Livingstone, Edinburgh.
- Friedberg, C. K., Cohen, H., and Donoso, E. (1968). Advanced heart block as a complication of acute myocardial infarction. Role of pacemaker therapy. Progress in Cardiovascular Disease, 10, 466.
- Lassers, B. W., and Julian, D. G. (1968). Artificial pacing in management of complete heart block complicating acute myocardial infarction. British Medical Journal, 2, 142.
- Lown, B. (1968). Discussion. In Acute Myocardial Infarction: Proceedings of a Symposium sponsored by the University of Edinburgh, Sept. 1967, p. 153. Ed. by D. G. Julian and M. F. Oliver. Livingstone, Edinburgh.
- Norris, R. M. (1969). Heartblock in posterior and anterior myocardial infarction. British Heart Journal, 31, 352.
- Sutton, R., Chatterjee, K., and Leatham, A. (1968). Heart-block following acute myocardial infarction treatment with demand and fixed-rate pacemakers. Lancet, 2, 645.
- Symposium on Acute Myocardial Infarction (1968). Discussion. In Proceedings of a Symposium sponsored by the University of Edinburgh, Sept. 1967, pp. 131-153. Ed. by D. G. Julian and M. F. Oliver. Livingstone, Edinburgh.